other values were compared. For each cross-section, the % LA increase (% LAI), and the distensibility (D = ΔLA/ΔPressure) in $mm^2/10^3$ mmHg was calculated. Results are shown in the table.

Conclusion: Remodeling affects the mechanical properties of the atherosclerotic arterial wall: shrunken arterial segments were more distensible than enlarged segments.

8:45

733-2 Time Course and Magnitude of Vascular Remodeling in Early and Advanced Coronary Atherosclerosis

M.H. Kim, S.E. Nissen, T. Crowe, K.M. Ziada, R.E. Hobbs, J.B. Young, E.M. Tuzcu. *The Cleveland Clinic Foundation, Cleveland, OH, USA*

Background and Objectives: The time course and magnitude of coronary remodeling (compensatory vessel enlargement) during the different stages of development of atherosclerosis remain unknown.

Methods: Patient population included 48 patients (103 segments) who underwent intravascular ultrasound within 4 weeks of cardiac transplantation (mild-moderate asymptomatic "donor" CAD) and 35 patients with symptomatic advanced CAD prior to intervention. Transplant recipients were divided into groups according to maximal plaque thickness. For both groups, a site with atherosclerotic plaque and an adjacent reference (least diseased) site were selected. Measurements included intimal thickness, area within external elastic membrane (EEM), lumen area, plaque area and EEM area. Area stenosis (%) and the ratio of lesion to reference EEM area (extent of compensatory enlargement) were calculated.

Results:	Mild-Moderate CAD			Advanced CAD
Plaque thickness (mm)	0.5–0.8	0.9–1.2	1.2–2.0	1.5–3.5
Plaque area (mm^2)	3.96	5.13	7.5	14.5
Lumen area (mm^2)	12.6	13.2	10.1	3.24
Lesion EEM area (mm^2)	16.6	18.3	17.6	17.7
Ref EEM Area (mm^2)	16.8	17.5	15.9	17.2
Lesion/Ref EEM ratio	0.99	1.06*	1.11	1.05
Area Stenosis (%)	25	30	44	82

*$p < 0.05$, $p < 0.005$ by ANOVA compared to least diseased group

Conclusions: Remodeling of the coronary arteries is not evident in the early stages of atherosclerosis (limited disease in asymptomatic transplant donors – mean stenosis 25%). Remodeling becomes more prominent with the increasing plaque area (more advanced disease in donors – mean stenosis 30–44%). Less remodeling is evident in symptomatic interventional patients with advanced disease (mean stenosis 82%), possibly reflecting impaired compensation.

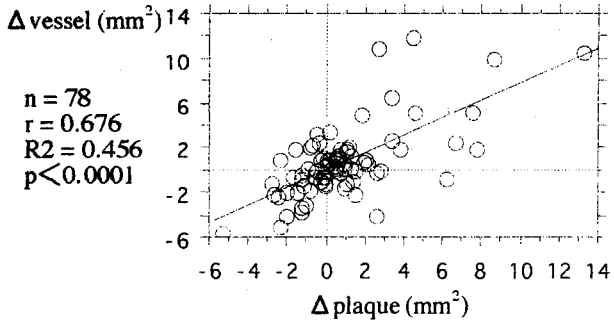
9:00

733-3 Compensatory Enlargement of Coronary Arteries after Directional Coronary Atherectomy

A. Kojima, T. Suzuki, H. Hosokawa, M. Yokoya, T. Suzuki. *National Toyohashi Higashi Hospital, Toyohashi, Japan*

To evaluate arterial changes after directional coronary atherectomy (DCA), we performed serial intravascular ultrasound in 78 lesions (70 patients), and measured vessel, lumen, and plaque areas of pre DCA, post DCA, and follow-up (6.18 ± 5.73 months). Δ vessel, Δ lumen, and Δ plaque were calculated.

Δ vessel (follow-up – post DCA)	0.65 ± 3.29 mm^2
Δ lumen (follow-up – post DCA)	−0.17 ± 2.51 mm^2
Δ plaque (follow-up – post DCA)	0.81 ± 2.86 mm^2



The correlation coefficient for Δ plaque and Δ vessel was significant (n = 78, r = 0.676, $p < 0.0001$), and regression analysis yield the following equation: Δ vessel (mm^2) = 0.777 × Δ plaque (mm^2) + 0.015.

Conclusion: We concluded that remodeling (local expansion) of vessels after DCA has occurred in close relation to Δ plaque and that this mechanism resulted in less severe compromise of the lumen than would be expected.

9:15

733-4 Doesn't Diseased Human Coronary Saphenous Vein Bypass Grafts Undergo Compensatory Enlargement? An Intravascular Ultrasound Study

J. Ge, G. Görge, M. Haude, F. Liu, D. Baumgart, V. Shah, S. Sack, R. Erbel. *Dept. of Cardiology, University Essen, Germany*

Coronary arterial remodeling is a common phenomenon in human atherosclerotic arteries. Controversies exist concerning the presence or absence of the remodeling process in diseased human coronary saphenous vein bypass grafts (SVBGs). The purpose of the study was to observe the vessel and lumen dimensions in patients who underwent SVBGs surgery with intravascular ultrasound (IVUS) to clarify whether the remodeling process exists in SVBGs. IVUS was performed in 43 SVBGs from 43 consecutive patients with a 3.5 F (Sonicath, Bonston Scientific Co.) or 3.2 F (MicroView, Boston Scientific Co.) 30 MHz IVUS catheter prior to any interventions. The vessel, lumen, and plaque areas were measured at the lesion segment, as well as the proximal and distal reference segments. The percent stenosis was calculated.

The vessel area in the lesion segment is significantly larger than that of the proximal reference segment ($p < 0.001$). It is also larger than that of the $\frac{1}{2}$ (proximal + distal) reference segment ($p < 0.001$). the vessel area increases in accordance with the increase of plaque area ($r = 0.94$, $p < 0.001$). In addition, a weak relationship was found between the vessel area and the percent stenosis increase ($r = 0.37$, $p = 0.04$).

Segments	Vessel area (mm^2)	Plaque area (mm^2)	Percent stenosis (%)
Proximal	12.8 ± 4.0	–	–
Lesion	19.0 ± 9.7	12.8 ± 7.6	64.5 ± 15.5
Distal	12.9 ± 3.6	–	–

In contrary to the previous finding, diseased SVBGs undergo focal compensatory enlargement (remodeling) in the presence of plaque formation. This remodeling process seems to be similar to the pathologic and IVUS findings in native coronary arteries.

9:30

733-5 Increased Restenosis in Diabetes Mellitus Following Transcatheter Coronary Interventions is Due to Exaggerated Intimal Hyperplasia

R. Kornowski, G.S. Mintz, J.J. Popma, K.M. Kent, A.D. Pichard, L.F. Satler, T.A. Bucher, A. Abizaid, R. Hoffmann, C.L. Walsh, M. Murphy, G. Bucher, M.B. Leon. *Washington Hospital Center, Washington DC, USA*

To understand why diabetic pts have an increased rate of restenosis, we compared serial (post-intervention and follow-up @ 5.6 mos) intravascular ultrasound studies in native vessel lesions in diabetic (DM, n = 60) vs non-diabetic pts (n = 162). Measurements included arterial, stent, lumen, and plaque (arterial-lumen) areas. Calculations included late lumen loss (Δ lumen area), intimal hyperplasia (stent-lumen area in stented lesions and Δ plaque area in nonstented lesions), and remodeling (Δ arterial area in nonstented lesions).

	DM	no DM	p
Stented lesions (n = 40)			
Late lumen loss (mm^2)	5.2 ± 2.5	2.0 ± 2.3	0.0009
Intimal hyperplasia (mm^2)	5.0 ± 2.8	1.8 ± 2.0	0.0007
Non-stented lesions (n = 182)			
Late lumen loss (mm^2)	3.2 ± 2.9	2.3 ± 3.9	0.1302
Intimal hyperplasia (mm^2)	1.3 ± 2.6	0.6 ± 2.5	0.0724
Remodeling (mm^2)	1.9 ± 2.8	1.8 ± 4.2	0.9093

Adaptive remodeling (late *increase* in arterial area) occurred in only 16% of diabetics compared to 28% of non-diabetics ($p = 0.0811$). *We conclude:* Serial intravascular ultrasound studies indicate that remodeling still plays an important role in DM (59% of late lumen loss). However, the reason for exaggerated late lumen loss and more frequent restenosis in diabetic pts is increased intimal hyperplasia, which is present in both stented and non-stented lesions.